

therapeutic regimens to orthopedic patients as to nonorthopedic patients without seriously weighing the risks of a dangerous increase in hemorrhage.

Many studies on the prevention of thromboembolism have been published during the last ten years—often with conflicting findings. Most deal with the incidence of deep vein thrombosis rather than the incidence of pulmonary embolism, yet venous thrombosis alone is of relatively minor consequence—its major importance is its relationship to the larger problem of pulmonary embolism. This relationship is still not fully understood.

The largest study on thromboembolism is the recently published international multicenter trial involving more than 4,000 general surgical patients, including 156 patients with total hip replacement. Its findings show an incidence of 0.7 percent fatal pulmonary embolism in patients receiving *no* prophylactic treatment. The incidence of fatal pulmonary embolism was reduced to 0.02 percent when subcutaneous heparin was given in the following way: 5,000 units every eight hours beginning two hours preoperatively and continuing for seven days or until the patient was ambulatory. However, there were *no* patients with acute fracture included in the multicenter trial, apparently for fear of hemorrhagic complications from the heparin. The patients undergoing total hip replacement who received heparin required 300 ml more blood than the untreated controls, and there was a slightly increased incidence of wound hematoma in the treated group as a whole.

These results are countered by those in a much smaller study which dealt exclusively with patients undergoing total hip replacement. Here it was shown that warfarin, low molecular weight dextran and aspirin were equally effective and all were superior to low dose subcutaneous heparin in decreasing the number of postoperative venous thrombi. With aspirin there was the added benefit of significantly fewer bleeding complications than with warfarin. The aspirin dosage schedule was a minimum of 600 mg the day before operation and postoperatively a minimum of 600 mg orally twice a day or 1,200 mg rectally twice a day until the patient was fully ambulatory and ready for discharge. The drug was discontinued abruptly at discharge.

Another modality now receiving attention is intraoperative intermittent calf compression. One attempt to assess the possible benefits of adding low dose heparin to the technique of intermittent

calf compression found that in 84 general surgical patients the prevention of deep vein thrombosis achieved by calf compression alone was not enhanced by the addition of low dose heparin.

The complete answer to prevention of pulmonary embolism is still elusive. At present, intraoperative mechanical intermittent calf compression, early postoperative ambulation and preoperative and postoperative aspirin prophylaxis have been shown to be effective in decreasing the incidence of thromboembolism in orthopedic patients without adding significant risks of hemorrhage.

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REFERENCES

- An international multicentre trial: Prevention of fatal post-operative pulmonary embolism by low doses of heparin. *Lancet* 2:45-51, Jul 12, 1975
- Harris WH, Salzman EW, et al: Comparison of warfarin, low-molecular-weight dextran, aspirin and subcutaneous heparin in prevention of venous thromboembolism following total hip replacement. *J Bone Joint Surg* 56A:1552-1562, Dec 1974
- Roberts VC, Cotton LT: Failure of low-dose heparin to improve efficacy of preoperative intermittent calf compression in preventing post-operative deep vein thrombosis. *Br Med J* 3:458-460, Aug 23, 1975

Tarsal Tunnel Syndrome

TARSAL TUNNEL SYNDROME is somewhat analogous to the carpal tunnel syndrome. It results from compression of the posterior tibial nerve within the tarsal canal. The tarsal tunnel lies behind the medial malleolus.

A patient with tarsal tunnel syndrome usually has complaint of burning pain in the plantar aspect of the foot, often in the metatarsal area. Occasionally the patient will note pain radiating up the medial side of the calf. The pain often is aggravated by activities and relieved somewhat by rest. Some patients, however, state that they have burning in their feet while in bed at night.

The onset of the syndrome usually is slow and insidious. In about half of such patients there is a history of some type of trauma.

Findings on physical examination will include a positive Tinel's sign over the area of the tarsal tunnel. The patient often will note that tapping the tarsal tunnel causes radiation of pain into the foot along the distribution of the medial or lateral plantar nerve. It is unusual to find any consistent motor weakness within the foot and only occasionally is there a sensory deficit.

The diagnosis of tarsal tunnel syndrome should be confirmed by electrodiagnostic studies. The studies should consist of nerve conduction times,

particularly the terminal latency of the medial plantar nerve to the abductor hallucis and the lateral plantar nerve to the abductor digiti quinti muscle. It should be emphasized that the latency period to both muscles should be determined since the syndrome may involve only one branch of the nerve. Electromyography also should be done to locate abnormal potentials (fibrillations). Both the abductor hallucis and abductor digiti quinti muscles again should be sampled.

When the diagnosis has been made and the clinical symptoms warrant it, surgical exploration with decompression of the tarsal canal and neurolysis of the posterior tibial nerve and its terminal branches should be carried out. In the decompression of the tarsal canal, extreme care must be taken not to disrupt the medial calcaneal branch which supplies sensation to the medial side of the heel.

In my experience there has been complete relief of symptoms in 80 percent of patients with this approach.

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REFERENCE

Mann RA: Tarsal tunnel syndrome. *Orthop Clin North Am* 5: 109-115, Jan 1974

and vascular compromise associated with tibial osteotomies.

In addition is *Group IV, miscellaneous*, which includes prolonged abnormal recumbent posture during drug induced stupor, snake bite, nephrosis, eclampsia, epilepsy, lumbar sympathectomy, disc surgery for low lumbar herniated nucleus pulposus, repair of muscle hernias and bracing.

Both acute and chronic forms of compartment syndromes do occur and usually require the same treatment unless foot drop has been complete for 10 to 12 hours. The simple anterior and lateral fasciotomy from the anterior approach is indicated if the anterior and lateral compartments only are involved but with fibulectomy and multiple fascial releases through its perostium if the posterior compartments are also involved. Early surgical release of compartment pressure is essential before irreversible muscle and nerve changes occur if satisfactory recovery is to result.

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REFERENCES

Bradley EL III: The anterior tibial compartment syndrome. *Surg Gynecol Obstet* 136:289, Jan 1973

Palmer BV, Mercer JL: Anterior tibial compartment syndrome following femoral artery perfusion. *Thorax* 28:492-494, July 1973

Wolfort FG, Mogelvong C, Filtzer HS: Anterior tibial compartment syndrome following muscle hernia repair. *Arch Surg* 106:97, Jan 1973

Compartment Syndromes of the Leg

COMPARTMENT SYNDROMES of the leg are recognized as resulting from an increasing number of diseases. Any mechanism that increases tissue pressure and decreases nutritional perfusion will reduce the microcirculation and may lead into a vicious circle resulting in ischemic necrosis. The anterior, lateral and deep posterior compartments of the leg are particularly vulnerable to such a process due to their confinement between bones and strong fascia. The superficial posterior compartment also may be involved particularly in crushing injuries.

Originally, clinical compartment syndromes were described as *Group I, idiopathic*, occurring most commonly in poorly conditioned persons subjected to great stress, such as young military recruits; and *Group II, traumatic*, associated with fractures and crushing injuries.

To this has been added *Group III, vascular*, associated with primary arterial or venous injury, or both. Group III now includes arterial perfusion which may be required as in open heart surgery

Blood Loss in Fractures of the Pelvis

MAJOR FRACTURES of the pelvis can cause life-threatening hemorrhage. The magnitude of blood loss is usually underestimated. Polytrauma is the rule, confusing the estimation of volume deficit and rate of depletion. Exsanguination under observation can be prevented only by very aggressive management.

A patient with a major arterial injury is seldom seen, but injuries to the small arteries can cause dangerously persistent bleeding. Because of abundant collaterals, hypogastric artery ligation has not been shown to be uniformly successful. Selective arteriography can help locate the injured vessel and may allow therapeutic clot or muscle embolization, or identify the site for ligation. However, most of the bleeding is of venous origin, and even with large vein laceration will subside with general measures and adequate immobilization. Since the pelvic fractures associated with hemorrhage are unstable, it is imperative that lower extremity traction be instituted promptly.